

Draw Pharmacology *in your mind*



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لا تتسوني من صالح دعائكم

Epilepsy

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Epilepsy

A group of **chronic** CNS disorders having in common the occurrence of sudden transient episodes (seizures) of:

1. Abnormal autonomic activity
2. Abnormal motor activity (convulsions)
3. Abnormal psychic activity

Etiology

- ◆ Epilepsy results from abnormal firing of a small population of neurons in some specific areas in brain → abnormal excitability → they are called "**primary focus**" or "**focal area**"
- ⊗ If the focal area is located in a **motor area** → epileptic seizures include **convulsions**
- ⊗ If located in a **parietal cortex** → seizures include **visual, auditory or olfactory hallucinations**

Classification

1. According to the cause

Primary epilepsy

- ◆ **No** anatomical cause, in newborn → brain malformations → lack or low oxygen during delivery
- ◆ Patient receives a **life-long** treatment

Secondary epilepsy

- ◆ There is an **anatomical cause** (ex. *trauma, Fever, Hypoglycemia, Meningeal infection*)
- ◆ Patient is treated **until** the cause is corrected

- ◆ The muscular activity is **limited to the same side** of the body and spread in an **orderly way** → ex. *when convulsions begin in fingers → hand → arm → leg → foot (all at the same side)*

- ◆ Patient is **conscious** during seizures (*awareness is retained*)

◆ Associated with

1. Impaired consciousness (*awareness is lost*)
2. Confused behavior
3. Loss of contact with environment (ex. can not even reply when someone talks to him)

1. Simple

2. Complex

- Affects *small portion* of the brain
- May progress to more complicated type (ex. *Generalized tonic-clonic seizures*)

Partial epilepsy

2. According to characteristics

- Begin as *focal seizures* (locally) then they spread to *both hemispheres* of the brain leading to abnormal electrical discharge
- May be convulsive or non-convulsive (*hallucinations as we said*)
- Immediate loss of consciousness
- The **attack** is very evident on EEG (*electroencephalogram*)

Generalized epilepsy

◆ Characterized by

1. Sensory **aura** (before the attack, the patient smells, sees or hears differently)
2. **Loss** of consciousness
3. Tonic contraction of all body muscles (muscle stiffness & rigidity)
4. Clonic spasm (repetitive jerking movements)
5. **Post-ectus sleep** (the patient sleeps after the attack)

3. Tonic-Clonic (Grand-mal)

4. Absence (Petit-mal)

- ◆ Occurs mostly in **children 4-12 years old**
- ◆ **Characterized by:** Sudden immobility - Vacant look in the eye - Eye-lid blinking
- ◆ Episode lasts for **30-60 seconds / more than 1** episode per day

5. Myoclonic

- ◆ **2ry epilepsy** occurs due to: Encephalitis - Drug poisoning - Uremia
- ◆ **Characterized by:** Short episodes of muscle contraction known as "**intermittent clonic jerks**"

6. Fibrile seizures

- ◆ **2ry epilepsy** occurs due to: **Fever** in **children** (3 months to 5 years old)

7. Status epilepticus

- ◆ Successive paroxysms نوبات متعاقبة of **tonic-clonic convulsions**
- ◆ So, it is a **severe, life-threatening** form of epilepsy → **emergency case**

Theories

- ◆ The initiation of epileptic attack begins from nerve cells in a **focal area**
- ◆ Those nerve cells differ from other nerve cells in their **unstable resting membrane potential**
- ◆ The **balance** between the excitatory neurotransmitter "**Glutamate**" and the inhibitory neurotransmitter "**GABA**" is **impaired** in the focal area

So treatment !

1. **Drugs stabilizing neuronal resting potential** → *they modify ionic conductance in nerve cells by:*
 - **Interfering with voltage-gated Na⁺ channels** ex. Phenytoin, Carbamazepine, Sodium valproate
 - **Interfering with Ca²⁺ channels** ex. Ethosuximide
2. **Drugs ↑ the effect of GABA** ex. Phenobarbital, Benzodiazepines, Gabapentine, Vigabatrin, Tiagabine, Progabide
3. **Drugs ↓ the effect of Glutamate** ex. Felbamate

Antiepileptics

1. Phenytoin



Mechanism

1. It **partially** blocks Na⁺ channels leading to:
 - Prolongation of refractory period
 - ↓ excitability of the membrane
 - ↑ the threshold of excitation
 → so it is a **membrane stabilizer**
2. It also
 - ↓ Ca²⁺ influx during depolarization
 - ↓ Glutamate release

Kinetics

- **Absorbed orally and extensively** metabolized by liver by **saturation kinetics** (*the molecules of the drug saturates the binding sites of the enzyme*) → so, the next doses will have **longer half-life** than the first one → may lead to **toxicity** if not well monitored
- **Dose** → once daily

Uses

- **All forms of epilepsy** except "**Absence seizures**" (*may worsen the case*)
- **Trigeminal neuralgia**

Adverse effects

1. Depression of CNS leading to **Nystagmus** تأرجح العين and **Ataxia**
2. **GIT disturbances** due to the alkalinity of the drug

Drug-Drug interaction

1. As it is **extensively metabolized** by liver → so it will be **affected by LME inducers and inhibitors**
2. As it is **LME inducer** → it will affect drugs metabolized by LME ex. **Praziquantel**
3. It is **highly protein bound** → so will compete with drugs bound to plasma proteins ex. **Valproate** (*LME inhibitor, Displaces phenytoin from plasma proteins*)



